Duke of Hazard

A Case of Uroabdomen in a Canine Patient

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Introduction:

Uroabdomen or uroperitoneum is the accumulation of urine within the abdominal cavity. Leakage of urine into the abdominal cavity can result from disruption of the urinary tract which include the kidneys, ureters, urinary bladder or proximal urethra. [2,3,5,7] Urinary bladder rupture is extremely common in veterinary patients and is the most common site of trauma within the urinary tract of dogs and cats. [2,3,6,7] There are no age or breed predispositions with urinary bladder ruptures, but it has been suggested that it occurs more frequently in males as their urethra is long, narrow and less-distensible as in females. [3,6] The most common causes of bladder rupture in the canine patient is blunt abdominal trauma and direct injury from pelvic fractures, but other causes reported in the literature include urethral obstruction, erosive neoplasms, iatrogenic rupture following cystocentesis, surgical complications, and penetrative wounds to the abdomen.[3,6,7,9]

All animals with a history of vehicular trauma should be assessed for urinary tract trauma.[3] Often these patients will not display clinical signs immediately following injury, so diagnosis may be delayed. Within 24-48 hours their condition may worsen as dehydration and severe metabolic/electrolyte imbalances may develop which can be fatal without appropriate treatment.[3,6] Nonspecific clinical signs will vary depending on duration and extent of uroabdomen and include, but are not limited to: vomiting, anorexia, depression, lethargy, reluctance to walk,

bradycardia, hematuria, stranguria, dysuria, abdominal pain, abdominal distention with or without a palpable fluid wave, as well as abdominal and/or perineal bruising.[1,2,3,5,6,7,10] It is important to note that if the rupture is small or located dorsally these animals may continue to have normal urination habits and fluid may still be retrievable during bladder catheterization.[2,3,5] These patients may also present in hypovolemic and/or septic shock with clinical signs of pale and tacky mucous membranes, tachycardia and weak peripheral pulses.[5,7] Cardiac dysrhythmias, respiratory distress, and altered mentation are also possible.[5,7]

A complete blood count, serum electrolyte concentration measurement, serum biochemistry profile, and acid-base status should be performed on all animals suspected of urinary bladder rupture if available. A diagnosis of uroabdomen should be considered when test results reveal azotemia, hyperkalemia, hyperphosphatemia, hypernatremia, metabolic acidosis, electrolyte derangements, increased hematocrit, and neutrophilia in conjunction with abdominal effusion confirmed on an abdominal focused assessment with sonography for trauma (A-FAST).[6,8,9,10] Plain radiographs may show loss of serosal detail causing the bladder to be undetectable and leaking of contrast from the urinary bladder can be observed with positive-contrast cystography.[2,6] An abdominocentesis should be performed to collect abdominal fluid which can be analyzed to compare abdominal fluid to peripheral blood creatinine and potassium ratios.[6,8,9,10] A definitive diagnosis of

uroabomen can be made when the creatinine ratio is ≥ 2:1 and potassium ratio of > 1.4:1.[6,8,9,10]

A prompt diagnosis of uroabdomen due to traumatic bladder rupture is crucial and aggressive emergency management of metabolic disturbances must take place to stabilize the patient followed by definitive surgical repair in most cases.[6] Delay in diagnosis and treatment can result in increased morbidity and mortality in these patients.[10] The prognosis for patients with traumatic bladder rupture is excellent in most cases as a high proportion of canine patients that receive appropriate emergency stabilization and surgical correction of the bladder survive to hospital discharge.[3,4,9]

History and Presentation:

Duke, an approximately 6-month old intact male white Great Pyrenees, presented to Mississippi State University College of Veterinary Medicine (MSU-CVM) Surgery Department as an emergency on May 3, 2018 for a suspected uroabdomen due to a ruptured bladder after being struck by a car at 2:00 pm on May 2, 2018. Duke originally presented to his referring veterinarian on May 2nd immediately following his injury. He was evaluated by a relief veterinarian who was concerned Duke may have a ruptured bladder due to his injury. A pneumocystogram was performed and radiographic findings were suspicious for uroabdomen. Duke was then referred to Mississippi State University College of

Veterinary Medicine (MSU-CVM) for further diagnostics and treatment. Duke's owners reported they took him home that evening because he seemed to be doing okay, but his status declined overnight, and his condition quickly worsened the morning of May 3rd. At this time the decision was made by Duke's owners to make the trip to MSU-CVM.

Duke presented to the Mississippi State College of Veterinary Medicine Surgery Department as an emergency on the afternoon of May 3rd, 2018. Upon presentation, he was severely depressed and laterally recumbent. He weighed 23.6 kg (51.9 lbs), had pale mucous membranes, a delayed capillary refill time of greater than 3 seconds (< 2 seconds), tachycardia at greater than 240 beats/minute (60 – 180 bpm), tachypnea at greater than 60 breaths/minute (10 – 40 brpm) and weak femoral pulses indicating Duke was in decompensated (hypodynamic) intravascular shock. He was hypersalivating and appeared extremely painful on palpation of his abdomen. A limited orthopedic examination did not reveal any injury to his bones. A murmur was auscultated which was suspected to be physiologic in nature. The remainder of Duke's physical examination was found to be within normal limits.

Diagnostic Approach/Considerations:

Upon Duke's arrival a blood sample was collected, and blood lactate was measured to be high at 5.6 mmol/L (<2.5 mmol/L) indicating poor vascular perfusion. An Azostix was completed revealing a result of 50-80 mg/dL (readings greater than 20 mg/dL warrant further investigation). Duke's packed cell volume (PCV) was 45% (35-55%) and his total protein was 9.5 g/dl (6.5-8.0 g/dL). A complete blood count, serum biochemical profile with electrolytes, arterial blood gas analysis, and coagulation profile was performed on Duke. The complete blood count was found to be unremarkable. The serum biochemical profile with electrolytes showed a severe hyperkalemia of 10.6 mmol/L (3.7-5.9 mmol/L), mild hypernatremia of 155.5 mmol/L (143-153 mmol/L), a markedly elevated BUN of 140 mg/dl (8-24 mg/dl) and creatinine of 6.18 mg/dl (0.5-1.4 mg/dl). The initial arterial blood gas analysis revealed a low SO2 of 90% (95-100%) and confirmed the presence of hyperlactemia, hypernatremia and hyperkalemia. Immediately following stabilization, another arterial blood gas analysis was performed showing a persistent hypernatremia, hyperkalemia, azotemia, and low SPO2 of 88%, but a normalized blood lactate of 0.9 mmol/L.

An abdominal focused assessment with sonography for trauma (A-FAST ultrasound) was performed on Duke which displayed free abdominal fluid in all four quadrants. An abdominocentesis was performed which revealed a cloudy, serosanguinous fluid which was submitted for analysis. Analysis of the abdominal

fluid revealed a creatinine measurement of 12.3 mg/dl which is almost 2 times the creatinine measurement of the blood (6.18 mg/dl) confirming the diagnosis of uroabdomen due to a ruptured bladder. A Jackson-Pratt peritoneal drain was placed to drain the abdomen of free fluid and a Foley indwelling urethral catheter was placed to assist with drainage of urine from the urinary bladder. Duke's SPO2 and blood pressure measurements were to be monitored every 4 hours. Continuous electrocardiogram (ECG) monitoring was continuously evaluated overnight to identify the presence of arrhythmias that may occur due to his electrolyte imbalance.

Once stabilized, abdominal and thoracic radiographs were performed on Duke. On interpretation of the abdominal radiographs, there was diffusely decreased serosal detail making complete evaluation of the liver, spleen and majority of the gastrointestinal tract difficult (peritoneal effusion) and the urinary bladder was unable to be definitively identified. The diaphragm remains intact and no obvious orthopedic abnormalities could be appreciated. Thoracic films were found to be within normal limits. Based on a recent history of trauma and diagnostic findings a confirmed diagnosis of uroabdomen due to traumatic bladder rupture was made.

Pathophysiology

The most common cause of uroabdomen in the dog is associated with vehicular or blunt trauma to the abdomen or pelvis with the most common site of rupture being the urinary bladder.[7,9] There is a direct correlation between the degree of bladder distention at the time of injury and probable bladder injury as the intraperitoneal pressure caused by the event results in a distended urinary bladder with stretched muscle fibers and a thin wall to rupture.[9] There is an increased risk of uroabdomen in patients who sustain pelvic fractures due to the deformation of the pelvic canal and sharp bone fragments lacerating the urinary bladder.[9]

Leakage of urine into the abdominal cavity allows nitrogenous waste products and electrolytes to be reabsorbed across the peritoneal membrane to reenter circulation.[3] Large molecules, like creatinine, diffuse slowly across the peritoneum to reenter circulation, therefore, creatinine remains concentrated within the free abdominal fluid resulting in abdominal fluid creatinine concentrations of greater than 2:1.[2,3,6,7,8] Small molecules, like potassium, rapidly equilibrate across the peritoneum into circulation resulting in hyperkalemia.[1,2,3,5,6] In an effort to maintain homeostasis, the body naturally responds by increasing renal excretion of potassium through effects of aldosterone and principal cells on Na+/K+ ATPase pumps in distal tubules and collecting ducts of the nephrons.[9] The rate at which potassium is excreted from the kidneys into the abdominal cavity exceeds the rate at which potassium is reabsorbed through the peritoneum into circulation resulting

in higher concentration of potassium in the abdominal fluid compared to the peripheral blood.[9] Comparing potassium levels within the abdominal fluid to serum concentrations can be a helpful diagnostic tool as an abdominal fluid-to-blood ratio greater than 1.4:1 is predictive of uroabdomen in canine patients.[1,3]

Complications can arise from increased serum potassium as hyperkalemia increases the resting membrane potential of cells within the body, including cardiac myocytes, resulting in an increase in cell membrane excitability.[9] Increased excitability within cardiac myocytes may result in potentially fatal cardiac arrhythmias, depending on the degree of hyperkalemia and coexisting abnormalities such as metabolic acidosis, progressing from bradycardia to premature ventricular complexes, ventricular fibrillation, and eventually asystole.[9]

Treatment and Management

Patients suspected of uroabdomen due to traumatic bladder rupture that are hyperkalemic and/or azotemic should first receive aggressive emergency stabilization to achieve correction of electrolytes, hydration and acid-base balance prior to surgery.[3,6] On presentation, Duke was in a state of decompensated intravascular shock in conjunction with hyperkalemia, azotemia, and hyperlactemia. An 18-gauge intravenous catheter was placed and Duke was administered an intravenous fluid bolus of Lactated Ringer Solution (LRS) at one-quarter of the total shock volume (90 mL/kg) and hetastarch in an effort to correct

electrolyte imbalances and restore normal intravascular fluid volume for appropriate perfusion. Since chemical peritonitis can be painful and because Duke was painful on palpation of his abdomen, he was administered intravenous methadone once as an analgesic on arrival.[6] An additional fluid bolus of LRS was administered and was then followed by a constant rate infusion (CRI) of LRS. This was continued overnight to further aid in the correction of electrolyte abnormalities, hydration deficit and acid-base imbalance.

Draining of the peritoneal effusion is essential in the management of electrolyte disturbances and azotemia in patients with traumatic bladder rupture.[6] Under local anesthetic, a Jackson-Pratt peritoneal drain was placed to relieve the abdomen of free fluid escaping from the ruptured urinary bladder.[6] Use of antibiotics may be warranted when urinary tract infections are suspected or prophylactically if abdominal drains have been placed.[3] Duke was administered Unasyn, a broad-spectrum antibiotic, intravenously every 8 hours to reduce the risk of infection that may accompany placement of the peritoneal drain. A Foley indwelling urethral catheter was placed to assist with drainage of newly formed urine from the urinary bladder which was emptied and quantified every 4 hours.[6] Duke was administered two puffs of Albuterol once. Albuterol is a beta-agonist that stimulates production of cyclic AMP through activation of adenyl cyclase and is predominantly a beta-2-agonist that causes relaxation of bronchial, uterine, and vascular smooth muscle,

but can also be used to promote a shift of potassium from the serum back into the cell resulting in temporary decreases in Dukes serum potassium levels.

Duke was found to be hypersalivating on his arrival indicating he was nauseated. As uremic gastritis can cause vomiting and ulceration he was placed on maropitant (Cerenia) and pantoprazole which were to be administered every 24 hours until signs resolved as gastrointestinal protectants.[6] Due to Duke's high respiratory rate and low oxygen saturation at presentation he was placed in an oxygen cage to provide supplementation of oxygen until he was determined to be able to sufficiently oxygenate on his own. For more sustained pain control, he was administered a bolus of fentanyl and was then started on a fentanyl CRI overnight to alleviate pain caused by leakage of urine within the abdomen. Overnight Duke began to display ventricular premature complexes (VPCs) in conjunction with tachycardia and pulse deficits, so he was administered a bolus of 2% lidocaine which was followed by a 2% lidocaine CRI to combat this cardiac arrhythmia. At Duke's 8:00 am assessment the following morning on May 4, 2018 a third blood gas analysis was performed to reveal an SO2 of 92%, a mild hypernatremia at 153.2 mmol/L, eukalemia and mild azotemia indicating he was making good progress.

Once patients with uroabdomen due to traumatic bladder are stabilized an exploratory laparotomy should be performed where rents in the bladder can be

identified, necrotic and damaged tissue debrided, and the bladder sutured with one or two layers of monofilament absorbable suture in an appositional suture pattern.[3,6] In cases where the bladder is thickened it is recommended that the bladder be closed with a single-layer appositional pattern.[3] Prior to routine closure of the abdomen, copious lavage of the peritoneal cavity should be performed and, if the integrity of the bladder wall is questionable, an indwelling urethral catheter should be placed for 24-48 hours for decompression.[6]

Following assessment and bloodwork at 8:00 am on Friday, May 4, 2018 (Day 2) Duke was determined to be stable and a surgical candidate to undergo an abdominal explore to assess his ruptured bladder. The 2% lidocaine CRI was discontinued, and he was prepped for an abdominal explore with an aseptic technique. A 25-cm incision was made on the ventral midline from the xiphoid process extending caudally to the pubis using a number 10 scalpel blade and the abdomen was entered routinely. The bladder was isolated, and a defect was located at the apex of the bladder which was used as a point of entry to drain the bladder of remaining urine. Exposed edges of the torn bladder tissue were trimmed away using Metzenbaum scissors and the bladder was closed using 3-0 PDS in a simple continuous suture pattern. A Foley urinary catheter was placed and infused with sterile saline to inspect the incision for leaks in which there were none appreciated. The Foley urinary catheter was removed, and the bladder was released into the

abdomen in a controlled manner. A total of 6 liters of warmed sterile saline were used to lavage the abdomen prior to closure. The abdomen was closed in a routine fashion and the skin was opposed with skin staples. A Jackson-Pratt peritoneal drain was placed and secured using Chinese finger trap sutures to ensure drainage of any remaining abdominal fluid. A Foley urinary catheter was placed to relieve the bladder of urine accumulation and allow appropriate healing.

Possible complications from a partial cystectomy include postsurgical urinary leakage if a watertight seal is not achieved and dehiscence.[3,6] There were no complications during Duke's partial cystectomy and he recovered well from anesthesia. Following surgery, he was continued on Unasyn, Plasmalyte A, and oxygen therapy and Duke was maintained on a fentanyl CRI that was adjusted as needed for management of postoperative pain until he was able to eat and drink on his own.

Following assessment on Saturday May 5, 2018 (Day 3) Duke was bright, alert and responsive. He had a good appetite, was oxygenating well on his own and determined to be adequately hydrated with ongoing intravenous fluid therapy. At this time, he was ambulatory and appeared to be lame in the left hind limb. A full orthopedic exam was performed with no abnormalities palpated or pain being located to any specific area, so the lameness was attributed to a possible muscle strain during his injury. Due to a healthy appetite, Duke was switched from

intravenous antibiotics, gastric protectants, and pain medication to oral Clavamox, Tylenol 4, and omeprazole respectively. He was started on a non-steroidal anti-inflammatory, carprofen, to assist in pain management and combat inflammation. On May 6, 2018 (Day 4) Duke's intravenous fluid therapy was discontinued as he was drinking well on his own. His Foley urinary catheter was pulled, and he was found to be urinating appropriately. At assessment on May 7, 2018 (Day 5) his Jackson-Pratt peritoneal drain was removed as minimal amounts of drainage was being collected. He was moved to the MSU-CVM Surgery ward where he continued to recover well with no complications until he was discharged on the morning of May 8, 2018 (Day 6).

Case Outcome:

At the time of his discharge Duke was bright, alert and responsive with a mild lameness observed in the left hind limb. He was discharged the morning of May 8, 2018 (Day 6) with carprofen, Tylenol 4, Clavamox, and omeprazole. His owners were advised to keep Duke confined to a kennel or small room with no furniture to ensure proper healing of his bladder and skin incision. If they noticed any excessive heat, swelling, pain, discharge or severe redness at his incision site it was recommended that he be seen by a veterinarian for evaluation of his incision. His owners were instructed to keep an Elizabethan collar on Duke at all times to prevent licking and biting of his incision which could result in serious

complications such as infection, suture failure, wound dehiscence or poor/delayed healing. It was recommended that he return in 14 days to evaluate his incision for appropriate healing, remove the skin staples and to explore his left hindlimb lameness should it remain unresolved.

Duke returned on May 17, 2018 for a staple removal in which his lameness was markedly improved. His owner mentioned he was urinating larger amounts followed by small amounts so it was recommended to Duke's owner that should he continue to urinate abnormally it is recommended that a urine culture be performed to rule out a urinary tract infection. I contacted Mrs. Hester on May 28, 2019 to get an update on Duke. She said he recovered well from his injury and has blossomed into the sweetest dog.

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